〈Regular Article〉

Kinesiological evaluation of respiration in patients with Parkinson's disease using optoelectronic plethysmography

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ABSTRACT Aspiration pneumonia, a common cause of mortality in patients with Parkinson's disease (PD), is associated with impaired coughing ability in addition to dysphagia. Kyphosis, bradykinesia, and muscle rigidity reportedly may be involved in the decline of coughing ability; however, the details are unknown. This study aimed to investigate the respiratory impairment that causes the decreased coughing ability in patients with PD using a three-dimensional motion analysis and surface electromyography in terms of respiratory pattern and respiratory muscle activity. The participants were seven patients with PD without history of respiratory or spinal diseases, and seven healthy men of age and matched for body mass index. Using optoelectronic plethysmography, the participants were placed in a sitting posture, and the volume changes of the upper and lower thorax and abdomen were measured during quiet breathing and during deep breathing in the same manner as in the measurement of lung capacity. Surface electromyograms (EMG) were recorded from the second intercostal muscle, the rectus abdominis muscle, and the external oblique abdominal muscle. The normalized EMG data were divided into inspiratory and expiratory times, and the inspiratory and expiratory times were divided into four equal parts over time to examine the trend of changes in muscle activity during deep breathing. In the patients with relatively unadvanced PD, no significant differences from the control in the amount of change in the angle of the spinal column during deep breathing, ratio of each compartment of the chest wall during deep breathing, whereas abdominal compartment was dominant in patients with PD during quiet breathing, and no significant difference in asynchrony between the chest and abdomen was observed. The PD group did not demonstrate increased respiratory muscle activity toward the end of inspiration and expiration, which was observed in the control group. Although the respiratory patterns of the thorax and abdomen in the PD group, whose disease has not relatively progressed, are not different from those of the control group, a significant difference was observed on the EMG, and it is possible that the respiratory muscles could not be effectively utilized according to the inspiratory and expiratory conditions from an early stage. doi：10.11482/KMJ-E202248119 *(Accepted on October 3, 2022)*

Key words：Parkinson's disease, Respiration, Optoelectronic plethysmography, Electromyography, Three-dimensional motion analysis system

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INTRODUCTION

Aspiration pneumonia accounts for approximately 24%-40% of deaths in patients with Parkinson's disease (PD)[1-3]. Meanwhile, dysphagia in patients with PD has been reported to be high at 50%-90%[4]. However, aspiration pneumonia is caused by impaired airway clearance in addition to dysphagia[5]. Bianchi *et al*. have reported that among patients with dysphagia with persistent aspiration, patients with pulmonary complications had significant lower mean cough peak flow (CPF) values[6].

PD is a progressive disease with gradual decline in motor function, in which, it is essential to maintain coughing ability for prevention of pneumonia. Cough consists of four phases: Phase 1 (induction), Phase 2 (inspiration), Phase 3 (compression), and Phase 4 (expiration)[7]. Insufficiency of any of these phases weakens cough, but sufficient inspiration is necessary as a prerequisite for utilizing laryngeal closure required in phase 3 and exhalation muscle strength in phase 4 to produce a strong cough. As the disease progresses, patients with PD are known to develop restrictive ventilatory impairment[8], but the details of the mechanism have not been investigated. Poor chest mobility and weakness of the respiratory muscles and kyphosis[9] as the secondary condition may cause insufficient inhalation and exhalation. Moreover, PD-specific symptoms such as bradykinesia, and muscle rigidity[10] may be involved in this process. We thought elucidating the factors of respiratory disorders would lead to effective pulmonary rehabilitation. Therefore, we decided to investigate the factors necessary to maintain lung capacity, such as thoracoabdominal respiratory movement and coordination, respiratory muscle activity, possible effects of bradykinesia and muscle rigidity.

Optoelectronic plethysmography (OEP) is a three-dimensional motion analysis system, which enables measurement of chest wall motion and volume, as well as that of the thorax and abdominal compartments. Florêncio *et al*. examined the breathing pattern during quiet breathing using OEP, but found no difference from healthy subjects[11]. However, deep breathing is necessary to maintain vital capacity, and we decided to investigate deep breathing in this study. Furthermore, the motion of the spine can be monitored simultaneously using the markers on the spine. Moreover, we considered applying the examination of rigidity in the extremities using surface electromyograms (EMG)[12] to the activity of the respiratory muscles in patients with PD. We thought that if there was respiratory muscle rigidity, myoelectric activity could be observed in the expiratory muscles during inspiration.

This study aimed to investigate the factors of respiratory disorders in patients with PD in terms of respiratory patterns and respiratory muscle activity to obtain useful knowledge for pulmonary rehabilitation using a three-dimensional motion analysis and surface EMG together.

MATERIALS AND METHODS

Participants

Male patients with PD aged 20 to 80 years (PD group) who were outpatients or inpatients of the Department of Neurology, Kawasaki Medical School Hospital between February 2019 and January 2022 and 60- to 80-year old healthy male participants (control group) were enrolled in this study.

The inclusion criteria were Hoehn-Yahr classification[13] of stage II, III, or IV in the PD group and ability to maintain a sitting position for approximately 30 min with upper limb support. The exclusion criteria were a history or presence of any of the following: head injury; cerebrovascular accident; spinal fracture; spinal cord injury; neurodegenerative diseases; neuromuscular diseases other than PD; fractures in the upper arm and

shoulder girdle; respiratory diseases such as chronic obstructive pulmonary disease and interstitial pneumonia, and insufficient treatment of PD (poor effect of oral medication, "on" period in on-off phenomenon cannot be maintained for 2-3 hours, involuntary movements which inhibit the sitting posture, or the presence of hallucinations and delusions).

Methods

Basic information and physical findings

In the PD group, the Hoehn-Yahr classification and MDS-UPDRS[14] motor examination was evaluated, and all of the following tests were performed during "on" periods.

Spirometry

Vital capacity (VC), % predicted VC (% VC), forced expiratory volume in one second ($FEV_{1.0}$), % predicted forced expiratory volume in one second (% $FEV_{1.0}$), and CPF were measured by spirometry in all participants (Autospiro AS-507, Minato Medical Science Co., Ltd., Osaka, Japan).

OEP

The participants were seated comfortably on a chair without a backrest with outerwear removed. Their shoulder joints were flexed and abducted by approximately 30°, and the forearms were placed on the upper limb platform. Referring to the method of Cala *et al*[15]., a total of 80 infrared reflection markers (markers) were placed on the body surface, comprising 35 on the front (chest and abdomen), 35 on the back, and 10 on the sides. A reference marker was placed on the right acromion. The marker horizon was placed through the jugular notch, sternal angle, papilla, xiphoid process, lower costal margin, umbilicus, and iliac crest. The vertical lines were drawn at the median, anterior, and posterior axillary lines, the midpoint between the median and anterior axillary lines, the midpoint between the median and posterior axillary lines, and the mid-axillary line (Fig. 1). The motion of all markers was recorded using eight infrared cameras (VERO, Vicon Motion Systems Ltd., Oxford, UK). All the participants performed deep breathing twice, which was same breathing as the VC measurement according to the Practical Handbook of Respiratory

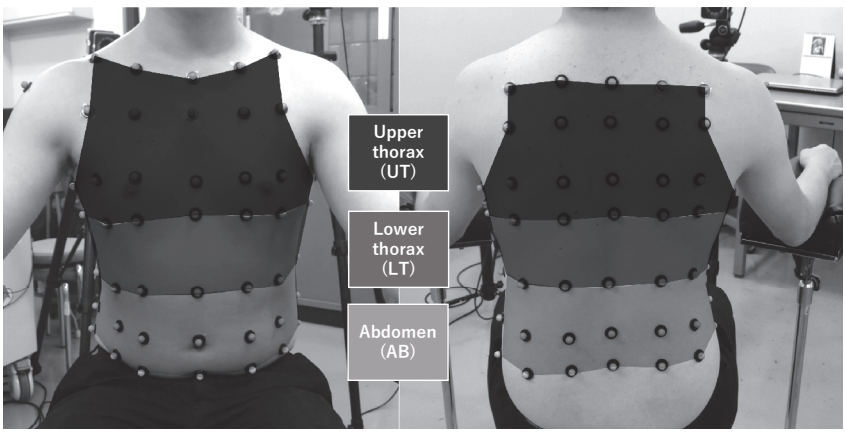

Fig. 1. Positions of the reflective markers on the chest wall and compartments
The marker horizon was placed through the jugular notch, sternal angle, papilla, xiphoid process, lower costal margin, umbilicus, and iliac crest. The vertical lines were drawn at the median, anterior, and posterior axillary lines, the midpoint between the median and anterior axillary lines, the midpoint between the median and posterior axillary lines, and the mid-axillary line. UT was the area superior to the xiphoid process, LT was the area from the xiphoid process to the lowest costal level, and AB was the area inferior to the lowest costal level.

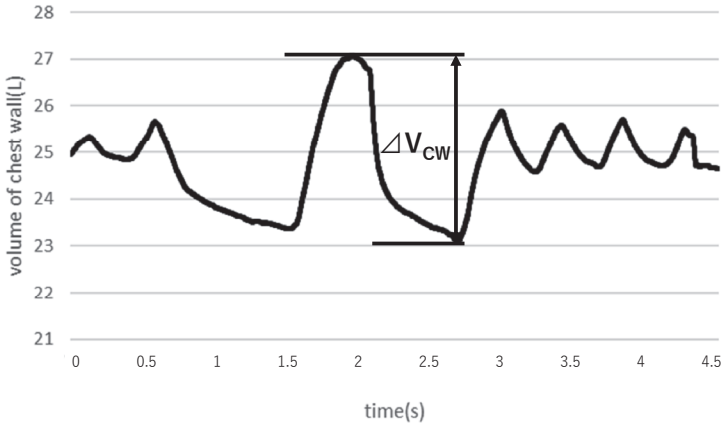

Fig. 2. Typical waveform of volume change over time of the chest wall
The ventilatory volume of each selected breathing was defined as the volume change during expiration.

Function Testing[16] with few minutes rest between each deep breathing after several quiet breaths. During the test, they were instructed to maintain their forearm on the upper limb platform. Each maker's motion during the test was recorded on a dedicated motion analysis software (NEXUS2, Vicon Motion Systems Ltd., Oxford, UK), and was then converted into coordinate data on the computer. The sampling frequency was 100 Hz.

Surface electromyography

The surface EMG was recorded at the same time as the OEP. Disposal surface electrodes (NCS electrode NM-31, Nihon Kohden Corporation, Tokyo, Japan) with a distance of 30 mm between the recording and reference electrodes were placed on the body surface on the right second intercostal muscle, right rectus abdominis muscle, and right external oblique muscle, according to the method of Perotto[17]. The ground electrode was placed on the upper right arm. The analog waveform obtained through the electromyogram (MEB-9104, Nihon Kohden Corporation, Tokyo, Japan) was converted into digital data by a motion analysis software (NEXUS2, Vicon Motion Systems Ltd., Oxford, UK) and recorded as Excel data on the computer. The bandpass filter was 30 Hz-500 Hz, and the sampling frequency was 1000 Hz.

Data analysis

OEP

The volume of the chest wall (V_{CW}) over time in each of quiet and deep breathing was calculated from the recorded coordinate data using a dedicated program on Matlab R2018a (The MathWorks, Inc., Natic, MA, USA).

The chest wall (V_{CW}) was divided into three compartments, the upper thorax, the lower thorax, and the abdomen (Fig. 1), and their respective volumes (V_{UT}, V_{LT}, and V_{AB}) were calculated. The method of dividing the chest wall into three parts was referred from that of Ferrigno *et al*[18].

The start and end points for each inspiration and expiration in each of quiet and deep breathing was defined as follows: the start of inspiration as the minimum value of V_{CW} immediately before inspiration, the end of inspiration as the maximum value of V_{CW} after inspiration, the start of expiration as the immediately after the end of inspiration, and the end of expiration as the minimum value of V_{CW} immediately before the next normal inspiration. The difference in volume from the inspiration start

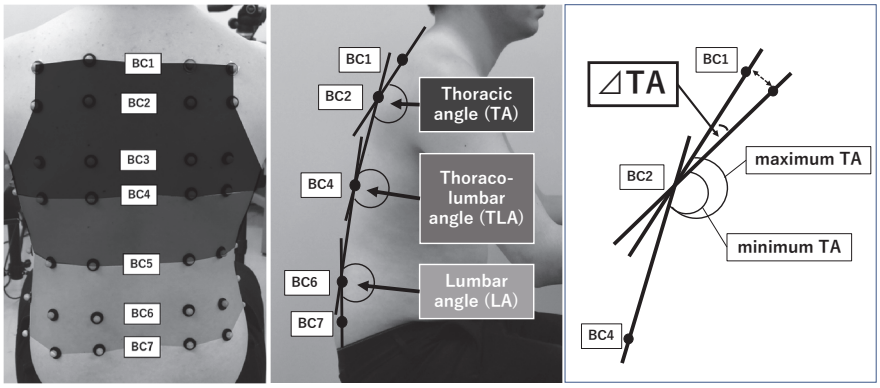

Fig. 3. The measurement of the amount of spinal angle changing during deep breathing
The amount of spinal angle changing during deep breathing was calculated using BC1, 2, 4, 6, and 7 markers which were placed on the spine. TA, TLA, and LA were calculated from the coordinates of each marker during deep breathing. The amount of change of each angle was calculated by subtracting minimum degree from maximum degree in each angle during deep breathing.

point to the expiration start point was defined as the inspiration volume change, and the difference in volume from the expiration start point to the expiration end point was the expiration volume change. In this study, the total amount of volume change in each breathing was defined as the volume change of the expiration (Fig. 2). The volume change was ΔV_{CW} for the entire chest wall, ΔV_{UT} for the upper thorax, ΔV_{LT} for the lower thorax, and ΔV_{AB} for the abdomen. The volume change of quiet breath in each participant was defined as calculating the mean of that of three breaths. Deep breathings were measured twice for each participant, and the one with the larger expiration ΔV_{CW} was adopted. To know the change of breathing pattern in the patient with PD, the ratio of the volume change of each compartment to ΔV_{CW} was calculated, and compared between the PD group and the control group.

Next, to know the effect of posture to the respiration, we measured the amount of change in the angle of the spine during deep breathing, using BC1, BC2, BC4, BC6, and BC7 markers placed on the spine, the angle formed by the markers BC1, 2, and 4 was defined as the thoracic angle (TA). The angle formed by markers BC2, 4, and 6 was defined as the thoracolumbar angle (TLA). The angle formed by markers BC4, 6, and 7 was defined as the lumbar angle (LA) (Fig. 3). The difference between the maximum and minimum values of each angle during the deep breath selected by volume measurement was calculated and defined as ΔTA, ΔTLA, and ΔLA (Fig. 3). The amount of change in spinal column angle during respiration was defined as $\Delta TA + \Delta TLA + \Delta LA$.

In addition, to know if paradoxical respiration in patient with PD is present, we examined the difference in the coordinated movement of both the chest and the abdomen during deep breathing between the PD and the control groups. The phase angle was calculated by creating the coordinated movements of the chest versus the abdomen plot for each subject.

Surface electromyography

To investigate the effect of abnormal muscle contraction in the patient with PD, we analyzed the EMG data during quiet and deep breathings. EMG data recorded at 1000Hz were integrated every 1/100 s. Integrated EMG (iEMG) data were normalized by calculating the percentage relative to the maximum value of iEMG data during selected

deep breathing. The normalized EMG data were divided into inspiration and expiration at the end point of inspiration of each selected breathing set by OEP. Typical waveform of temporal change of volume in chest wall and iEMG during deep breathing in patient with PD and control are shown (Fig. 4).

In quiet breathing, normalized EMG data integrated by the sequence for each of inspiration and expiration was defined as the amount of myoelectricity in each of them. The mean values of the amount of myoelectricity in each of inspiratory and expiratory phase were compared between PD group and control group.

In deep breathing, the inspiratory time and expiratory phase were divided into four equal parts: section 1, section 2, section 3, and section 4 (Fig. 5). Normalized EMG data integrated by the sequence for each section was defined as the amount of myoelectricity in that section. The amount of

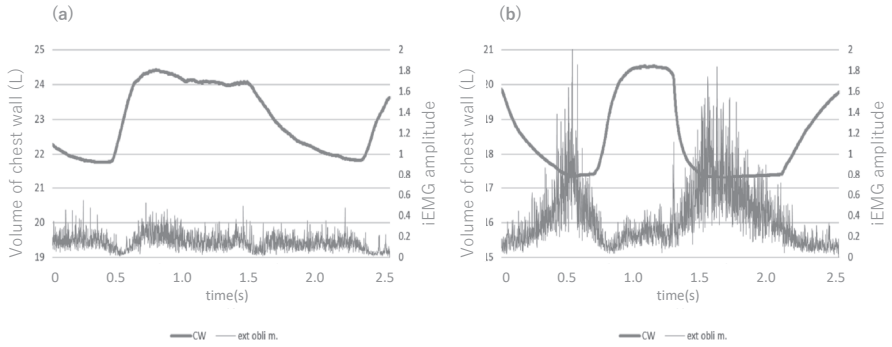

Fig. 4. Typical waveform of temporal change of volume in chest wall and iEMG during deep breathing in patient with Parkinson's disease and control
(a) Patient with PD, (b) control; Thick line shows the waveform of temporal change of volume in chest wall (left axis), and thin line shows integrated EMG on right external oblique muscle (right axis).

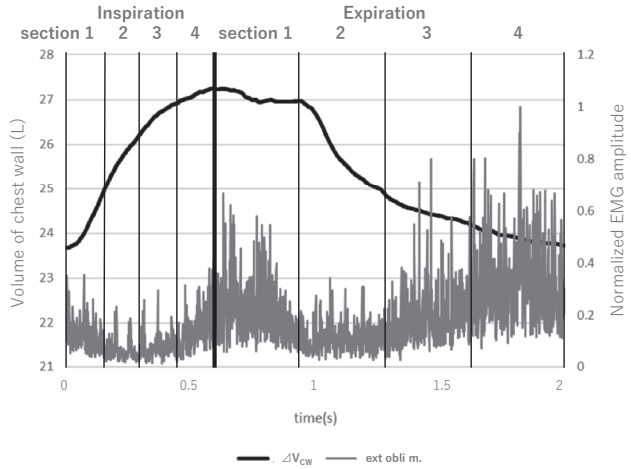

Fig. 5. Typical waveform of temporal change of volume in the chest wall and normalized EMG during deep breathing in control
The thick line shows the temporal change of volume in the chest wall (left axis), and thin line shows normalized EMG (right axis).
The inspiration and expiration phases were respectively divided into four equal sections (dotted lines).

myoelectricity in each section was calculated for each muscle in all the participants. The mean values of the amount of myoelectricity for each muscle in each participant was compared among sections.

Statistical analyses

SPSS version 25 (IBM Corporation, Armonk, NY, USA) was used for all statistical analyses, and an unpaired T-test was used for comparison between groups, with a confidence interval of 95% and a significance level of 0.05. The Jonckheere-Terpstra test was used to examine the tendency of the mean value of myoelectricity in each muscle for each group.

This study was approved by the Institutional Review Board of Kawasaki Medical School Hospital (approval number 3359-03). This research was supported by a Grant-in-Aid for Scientific Research (Research Leader: Kozo Hanayama, Research Project/Area Number: 19K11336). Written informed consent was obtained from all the participants in accordance with the Declaration of Helsinki.

RESULTS

No significant differences in age and BMI were observed between the two groups. The spirometry results indicated that VC and CPF were significantly lower in the PD group than in the control group. Meanwhile, %VC, $FEV_{1.0}$%, and $\%FEV_{1.0}$ did not significantly differ between the two groups. No significant difference in the change in spine angle during deep breathing was observed between the two groups (Table 1). The severity of PD in the patients was II in five, III in one, and IV in one on the Hoehn-Yahr scale (Table 2).

We calculated each fraction set as a percentage of ΔV_{CW} during quiet breathing. ΔV_{UT} was 30.3 ± 11.4%, ΔV_{LT} = 22.7 ± 10.5%, and ΔV_{AB} = 47.1 ± 13.4% in the PD group, respectively. In the control group, ΔV_{UT} = 42.5 ± 9.6%, ΔV_{LT} = 23.6 ± 8.8%, and ΔV_{AB} = 33.6 ± 10.9%. The control group had a significantly greater proportion of the upper thorax than the PD group (p = 0.001), while the PD

Table 1. Patient data, respiratory function test, and change of spinal angle in the patient and control groups.

	Mean ± SD		p-value
	Patients (N = 7)	Control (N = 7)	
Age (years)	71.0 ± 9.1	64.6 ± 4.0	0.114
BMI (kg/m^2)	24.0 ± 3.3	23.0 ± 3.1	0.560
VC (L)	3.0 ± 0.5	3.8 ± 0.5	0.014*
%VC (%)	83.6 ± 9.5	95.4 ± 12.0	0.063
$FEV_{1.0}$% (%)	78.8 ± 4.1	79.7 ± 5.7	0.746
$\%FEV_{1.0}$ (%)	98.3 ± 5.1	97.6 ± 6.3	0.820
CPF (L/min)	425.7 ± 98.3	572.9 ± 88.3	0.012*
Change in spine angle during deep breathing (degree)	12.8 ± 6.9	14.4 ± 2.5	0.589

Patient data, spirometry, and spine angle during deep breathing are presented. VC and CPF were significantly smaller in the patient group.
BMI, body mass index; VC, vital capacity; FEV, forced expiratory volume; CPF, cough peak flow.
* $p < .05$ ** $p < .01$

Table 2. Profile data of the enrolled patients with Parkinson's disease.

Severity (Hoehn and Yahr scale)	Age	BMI	UPDRS motor	VC	%VC	$FEV_{1.0}$%	$\%FEV_{1.0}$	FVC	%FVC	CPF
Ⅱ	69	29.0	17	3.7	97	81.1	100	3.3	89	600
Ⅱ	53	21.5	26	3.8	86	78.9	94	3.7	88	350
Ⅱ	79	22.5	38	2.5	69	81.6	104	2.6	75	380
Ⅱ	67	22.3	13	2.9	87	82.4	102	3.0	89	500
Ⅳ	77	28.4	17	2.6	86	78.3	99	2.3	78	340
Ⅲ	74	21.2	1	2.9	87	79.4	100	2.2	70	460
Ⅱ	78	23.0	18	2.6	73	70.2	89	2.4	70	350

The severity of PD was II in five patients, III in one patient, and IV in one patient on the Hoehn and Yahr scale. % VC was < 80% in 2 of 7 patients, and FEV 1.0% was < 70% in none.
BMI, body mass index; UPDRS, unified Parkinson's disease rating scale; VC, vital capacity; FEV, forced expiratory volume; FVC, forced vital capacity; CPF, cough peak flow.

Table 3. Volume change of each compartments of the chest wall during quiet breathing.

	Mean ± SD		P-value
	Patients (N = 7)	Control (N = 7)	
ΔV_{UT} (%)	30.3 ± 11.4	42.5 ± 9.6	0.001**
ΔV_{LT} (%)	22.7 ± 10.5	23.6 ± 8.8	0.700
ΔV_{AB} (%)	47.1 ± 13.4	33.6 ± 10.9	0.001**

The ratios of ΔV_{UT}, ΔV_{LT}, and ΔV_{AB} to ΔV_{CW} are presented. During quiet breathing, the control group had a significantly greater proportion of the upper thorax than the PD group (p = 0.001), while the PD group had a significantly greater proportion of the abdomen than the control group (p = 0.001).
* p < .05 ** p < .01

Table 4. Volume change of each compartments of the chest wall during deep breathing

	Mean ± SD		P-value
	Patients (N = 7)	Control (N = 7)	
ΔV_{UT} (%)	38.5 ± 9.5	37.0 ± 8.1	0.758
ΔV_{LT} (%)	26.7 ± 5.6	27.4 ± 6.7	0.840
ΔV_{AB} (%)	34.8 ± 11.7	35.6 ± 9.8	0.892

The ratios of ΔV_{UT}, ΔV_{LT}, and ΔV_{AB} to ΔV_{CW} are presented. No significant difference in each volume change was observed between the two groups.

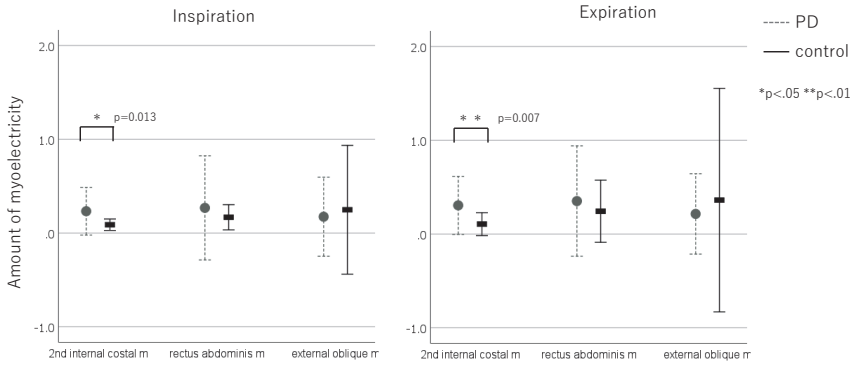

Fig. 6. Mean values of the amount of myoelectricity for each muscle during quiet breathing
The mean value with 1SD of the amount of myoelectricity for each muscle in PD and control groups during quiet breathing are shown by error bar graph. Thin dotted line shows that of PD group, and thick line shows that of control group.

group had a significantly greater proportion of the abdomen than the control group (p = 0.001) (Table 3).

And when ΔV_{CW} during deep breathing was set to 100%, the proportions of each fraction were ΔV_{UT} = 38.5 ± 9.5%, ΔV_{LT} = 26.7 ± 5.6%, and ΔV_{AB} = 34.8 ± 11.7% in the PD group, respectively. In the control group, ΔV_{UT} = 37.0 ± 8.1%, ΔV_{LT} = 27.4 ± 6.7%, and ΔV_{AB} = 35.6 ± 9.8%. No significant difference in each volume change was observed between the two groups (Table 4).

The phase angle was 12.5 ± 13.1° in the PD group and 5.9 ± 17.3° in the control group (p = 0.433), indicating no significant difference between the two groups.

During quiet breathing, the mean values of amount of myoelectricity of the second intercostal muscle in the PD group was significantly greater than that of the control group during both inspiration (p = 0.013) and expiration (p = 0.007) phases. In other muscles, there was no significant differences in the mean values of amount of myoelectricity between the two groups (Fig. 6).

During deep breathing, the PD group showed no obvious change in EMG from interval 1 to interval 4 in both the inspiratory and expiratory phases. On the other hand, in the control group, the myoelectricity gradually increased over section 4 in the second intercostal muscle of the inspiratory phase, the rectus abdominis and the external oblique

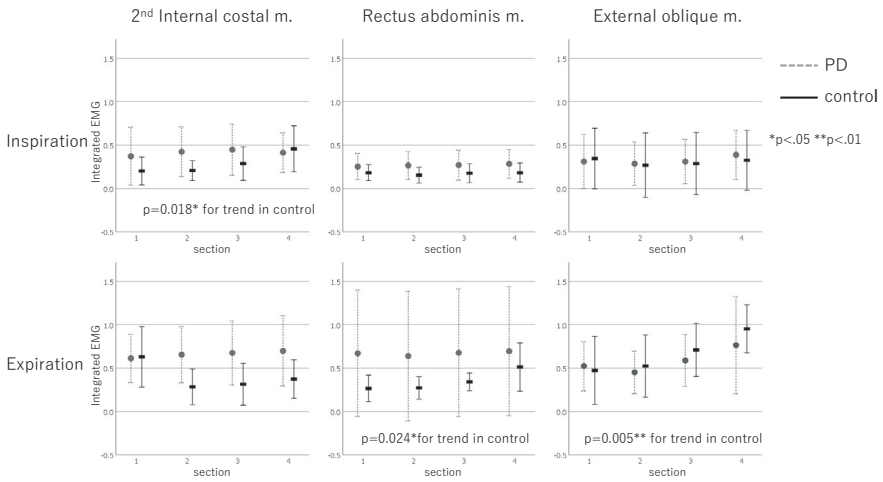

Fig. 7. Mean values of the amount of myoelectricity in each section for each muscle during deep breathing
The mean value with 1SD of the amount of myoelectricity in each sections during deep breathing is shown by error bar graph for each PD and control groups.

muscles of the expiratory phase. In the tendency analysis, no significantly increasing or decreasing trend in the mean value of amount of myoelectricity was observed in both the inspiratory phase and expiratory phase in the PD group. In the control group, the amount of myoelectricity tended to increase from section 1 to section 4 in the second intercostal muscle of the inspiratory phase ($p = 0.018$). The rectus abdominis muscle ($p = 0.024$) and the external oblique muscle ($p = 0.005$) in the expiratory phase also tended to increase in myoelectricity from section 1 to section 4 (Fig. 7).

DISCUSSION

Five out of 7 patients with PD in this study were had classified into the Hoehn-Yahr stage Ⅱ and in good control. In such group of patients, significant differences were observed in the rate of change in the volume of each compartment of the chest wall during quiet breathing, with a greater rate of change in the abdomen in the PD group than in the control group, but no significant differences were observed compared with the control in the amount of change in the angle of the spinal column during deep breathing, volume change ratio of each compartment of the chest wall during deep breathing, and thoracoabdominal asynchrony.

This study showed abdominal predominance during quiet breathing in patients with PD, which has not reported before. This is because the ribcage may be relatively stiffer than the abdomen in PD patients, however, both thoracic and abdominal breathing are seen in healthy subjects, and it is unclear whether this study is meaningful because of the small number of cases. But this pattern diminished during deep breathing. In deep breathing, we found no significant difference of fractionation compared with that in healthy subjects.

The participants were often observed to move the trunk back and forth during deep inhalation and exhalation during the spirometry test. Since the trunk movement decreases in patients with PD, we hypothesized that insufficient trunk movement during deep breathing might be the cause of the decrease in VC. However, no significant difference in the amount of change in the angle of the spinal

column during a deep breath was observed. No relationship between trunk movement and respiratory function was identified. Since the participants kept fixed both shoulder joints to the upper limb base by flexion/abduction during measurement to avoid situations, where the marker of the lateral thorax and axilla is not visible from any camera, and the movement of the spinal column may be limited. The influence of the posture adopted in this study may have appeared because the volume change of the upper and lower thorax was underestimated by the posture with both upper limbs raised during OEP measurement which increases the amount of functional residual capacity[19].

In a report examining the chest wall movement patterns of patients with PD, Vercueli *et al.* used plethysmography to analyze resting compartmental breathing patterns of the chest wall in patients with PD and observed that the patterns were different when levodopa was on the "on" stage compared with the "off" stage, suggesting that rigidity and dyskinesia may be involved in respiratory movements in PD[20].

Several studies have reported on the rigidity on the extremities, i.e. quantitative measurement methods using accelerometers, angular velocity meters, and surface electromyograms[12]. However, investigating muscle rigidity in the respiratory muscles using the same method is difficult. Rigidity is a muscle phenomenon that occurs when a muscle is passively stretched, and its degree depends on the stretch rate and amplitude[21]. Thus, whether it can be observed with small muscles such as the respiratory muscles remains unclear. This may be a finding of muscle rigidity in the respiratory muscles of a patient with PD, or of extra muscle activity for maintaining posture. In Jaroslaw *et al.*'s study, patients with PD and healthy controls held a two-kilogram load in an upright position, and the surface EMG in the biceps brachii and triceps brachii muscles were measured. Accordingly, a 6-Hz burst, which was not observed in healthy participants, was recorded in the leading and antagonistic muscles in patients with PD, suggesting the possibility of a neuromuscular degenerative change in patients with PD[22]. Using an electromyogram to search for the influence of rigidity is considered effective; however, the activity of the trunk muscles is affected by the posture[23, 24]. Thus, relationship between the posture and muscle activity should be carefully considered. In this study, it was difficult to demonstrate the effect of EMG on muscle rigidity. However, the PD group lost the characteristic features of respiratory muscle activity toward the end of inspiration and expiration seen in the control group, suggesting that the chest wall movements used during inspiration and expiration were impaired. The findings obtained in this study could be utilized when instructing breathing exercise in patients with PD.

The limitations of this study were the resulting low severity and bias of patients with PD and the small number of cases. In the future, more detailed studies investigating patients with PD with a greater range of severity and a larger sample size will be needed to investigate whether the respiratory pattern changes or the thoracic mobility decreases as the severity progresses. Moreover, examining whether the findings of muscle activities in this study can be utilized for respiratory muscle training is necessary to prevent aspiration pneumonia in patients with PD.

CONCLUSION

Although the respiratory patterns of the chest and abdomen in patients with PD, whose disease have not relatively progressed, are not different from those of the control, a significant difference was observed on the EMG, and the respiratory muscles could not be effectively utilized according to the inspiratory and expiratory conditions from an early stage. Effective respiratory muscle training methods should be developed for applying from an early

stage in patients with PD.

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CONFLICTS OF INTEREST

There are no conflicts of interest to declare in this study.

REFERENCES

1）Nakashima K, Maeda M, Tabata M, Adachi Y, Kusumi M, Ohshiro H: Prognosis of Parkinson's disease in Japan. Tottori University Parkinson's Disease Epidemiology (TUPDE) Study Group. Eur Neurol. 1997; 38: 60-63. doi: 10.1159/000113485.

2）Beyer MK, Herlofson K, Arsland D, Larsen JP: Causes of death in a community-based study of Parkinson's disease. Acta Neurol Scand. 2001; 103: 7-11. doi: 10.1034/j.1600-0404.2001.00191.x.

3）Fall PA, Saleh A, Fredrickson M, Olsson JE, Granérus AK: Survival time, mortality, and cause of death in elderly patients with Parkinson's disease: a 9-year follow-up. Mov Disord. 2003; 18: 1312-1316. doi: 10.1002/mds.10537.

4）Leopold NA, Kagel MC: Laryngeal deglutition movement in Parkinson's disease. Neurology. 1997; 48: 373-376. doi: 10.1212/wnl.48.2.373.

5）Hasegawa K, Moroboshi T, Fukui T, Kawamura M, Sugita K: Examination of factors involved in the prognosis of Parkinson's disease. Showa Univ J Med Sci. 1997; 57: 69-78.

6）Bianchi C, Baiardi P, Khirani S, Cantarella G: Cough peak flow as a predictor of pulmonary morbidity in patients with dysphagia. Am J Phys Med Rehabil. 2012; 91: 783-788. doi: 10.1097/PHM.0b013e3182556701.

7）Myslinski MJ, Scanlan CL: Bronchial Hygiene Therapy. In: Fundamentals of respiratory care, 9th ed. (Wilkins RL, Stroller JK, Kacmarek RM eds). St. Louis, Mosby. 2009; 921-923.

8）Guilherme EM, Moreira RFC, de Oliveira A, Ferro AM, Di Lorenzo VAP, Gianlorenço ACL: Respiratory Disorders in Parkinson's Disease. J Parkinsons Dis. 2021; 11: 993-1010. doi: 10.3233/JPD-212565.

9）Nakayasu T, Yamamoto S: Effect of kyphosis on thoracic movement. Journal of Physical Therapy Science. 2009; 24: 697-701.

10）Santos RBD, Fraga AS, Coriolano MDGWS, Tiburtino BF, Lins OG, Esteves ACF, Asano NMJ: Respiratory muscle strength and lung function in the stages of Parkinson's disease. J Bras Pneumol. 2019; 45: e20180148. doi: 10.1590/1806-3713/e20180148.

11）Florêncio RB, da Nobrega AJS, Lima INDF, Gualdi LP, Cabral EE, Fagundes MLLC, Aliverti A, Resqueti VR, Fregonezi GAF: Chest wall volume and asynchrony in stroke and Parkinson's disease subjects: A case-control study. PLoS One. 2019; 14: e0216641. doi: 10.1371/journal.pone.0216641.

12）Ferreira-Sánchez MDR, Moreno-Verdú M, Cano-de-la-Cuerda R: Quantitative Measurement of Rigidity in Parkinson's Disease: A Systematic Review. Sensors (Basel). 2020; 20: 880. doi: 10.3390/s20030880.

13）Hoehn MM, Yahr MD: Parkinsonism: onset, progression and mortality. Neurology. 1967; 17: 427-442. doi: 10.1212/wnl.17.5.427.

14）Goetz CG, Tilley BC, Shaftman SR, *et al.*: Movement Disorder Society-sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS): scale presentation and clinimetric testing results. Mov Disord. 2008; 23: 2129-2170. doi: 10.1002/mds.22340.

15）Cala SJ, Kenyon CM, Ferrigno G, Carnevali P, Aliverti A, Pedotti A, Macklem PT, Rochester DF: Chest wall and lung volume estimation by optical reflectance motion analysis. J Appl Physiol. 1996; 81: 2680-2689. doi:10.1152/jappl.1996.81.6.2680.

16）Japanese Respiratory Society Pulmonary Physiology Committee Practical Handbook of Respiratory Function Testing preparation committee: Practical Handbook of Respiratory Function Testing. Tokyo, Medical Review Co., Ltd.2021.

17）Perotto AO (Translated by Kayamori R): Anatomical Guide for the Electromyographer: The Limbs and Trunk (3rd edition). Tokyo, Nishimura Co., Ltd. 1997.

18）Ferrigno G, Carnevali P, Aliverti A, Molteni F, Beulcke G, Pedotti A: Three-dimensional optical analysis of chest wall motion. J Appl Physiol. 1994; 77: 1224-1231. doi: 10.1152/jappl.1994.77.3.1224.

19）Martinez FJ, Vogel PD, Dupont DN, Stanopoulos I, Gray A, Beamis JF: Supported arm exercise vs unsupported arm exercise in the rehabilitation of patients with severe

chronic airflow obstruction. Chest. 1993; 103: 1397-1402. doi: 10.1378/chest.103.5.1397.

20) Vercueil L, Linard JP, Wuyam B, Pollak P, Benchetrit G: Breathing pattern in patients with Parkinson's disease. Respir Physiol. 1999; 118: 163-172. doi: 10.1016/s0034-5687(99)00075-4.

21) Powell D, Threlkeld AJ, Fang X, Muthumani A, Xia R: Amplitude- and velocity-dependency of rigidity measured at the wrist in Parkinson's disease. Clin Neurophysiol. 2012; 123: 764-773. doi: 10.1016/j.clinph.2011.08.004.

22) Marusiak J, Jaskólska A, Kisiel-Sajewicz K, Yue GH, Jaskólski A: EMG and MMG activities of agonist and antagonist muscles in Parkinson's disease patients during absolute submaximal load holding. J Electromyogr Kinesiol. 2009; 19: 903-914. doi: 10.1016/j.jelekin.2008.03.003.

23) Kera T, Maruyama H: The effect of posture on respiratory activity of the abdominal muscles. J Physiol Anthropol Appl Human Sci. 2005; 24: 259-265. doi: 10.2114/jpa.24.259.

24) Tomita Y, Suzuki Y, Tanaka Y, Hasegawa Y, Yoshihara Y, Okura K, Matsuka Y: Effects of sitting posture and jaw clenching on neck and trunk muscle activities during typing. J Oral Rehabil. 2021; 48: 568-574. doi: 10.1111/joor.13152.